

Villous Stromal Hemorrhage in Placental Villi during Chronic Hypertension

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Abstract

With the purpose of to describe the vessels of the placental villi in hemorrhage condition during chronic hypertension associated to obesity two placentas were analyzed with light microscopy. Stem villi were observed with closed or opened vessels in their trajectory. Numerous intermediate villi were noted with dilated vessels that refill the stromal region. Remarkable changes as picnotic nucleus, nuclear disintegration, thinness and interruption of the syncytium were found. The vessels have exploited in the stromal region. Syncytium very thin surround to numerous erythrocytes. A disorganized structure of the stem villi with frequency can be seen. These villi have a great deal coverture of fibrinoid deposition and others are observed buried by fibrinoid. Chronic hypertension has provoked intravillous hemorrhage in the placental villi which could to put in danger the fetal good development with decreased fetal nutrition.

Keywords

Placental hemorrhagic; Villi; Obesity; Chronic hypertension

Introduction

Intravillous hemorrhage in placental villi has been seen in preterm placentas associated to suspected abruption often accompanying sudden anoxia [1]. This event provokes early mortality in preterm infants and is likely due to sudden ischemia, hemodynamic shifts within the villous circulation, leading to the rupture of capillary walls and vascular instability in very low birth weight infants [2].

This feto-maternal hemorrhage has been classified as fetomaternal hemorrhage of small vessel type and fetomaternal hemorrhage of large vessel type [3]. Accumulations of clotted blood in the intervillous space or intervillous thrombus represent foci of fetomaternal hemorrhage clinically important when are multiple or very large [4]. These represent the first type.

Rupture of large fetal vessels in the chronic plate or larger stem villi provoke a massive subchorial thrombosis that elevates the chronic plate while displacing the under lying villous parenchyma downward and is associated with preterm intrauterine fetal death [5]. This event is a catastrophic and random contingency representing the second type. If well it has been described that "whether stromal hemorrhage is seen in situations other that abruption is not clear" [3]. Nevertheless, we have observed the rupture of vessels in the stromal region of the placental villi in patients with chronic hypertension. To describe the placental villi with the vessels in hemorrhagic condition.

The conditions leading to friability of terminal villi such as increased syncytial knotting in preterm infants and delayed villous maturation in term infants may increase the risk of villous rupture [3].

The fetus often bleeds into the maternal circulation for many reasons as trauma to the placenta, therapeutic abortion, caesarean section, automobile accidents, fetal kicking, turning or punching. Factors that can provoke exsanguination and may cause severe fetal anaemia, mortality and morbidity [6].

Is our objective to describe the vessels of the placental villi in hemorrhagic condition during chronic hypertension associated to obesity?

Case Study

Two placentas obtained by cesarean of woman pregnancy at 32 (P1) and 39 (P2) years old were processed by light microscopy and H&E stain. Patient P1 with history of two cesarean, one abortion and 117 kg of weight while that patient P2 had 67kg of weight without that history. Both patients without pregnancy induced hypertension have given their informed consent for participation in the research study and there is not conflict of interest in this work. Five fragments of placenta were selected by each placenta and compared with normal placenta as control. Four slides prepared by each fragment and 20 slides by placenta were examined. In this work observations were done in hemorrhagic

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zones of placental villi whose patients had hypertension considered $\geq 140/190$ mmHg on two occasions that presents before 20 weeks gestation and not come back to normal within 12 weeks of delivery.

Results

Stem villi were observed with closed vessels, associated with degenerative villi which contain clear regions in their stromal region (Figure 1). In others the stem villi presented an opened vessels and another closed vessel in their trajectory (Figure 2). Numerous placental villi as mature intermediate villi were noted with dilated vessels that refill the stromal region (Figure 3).

The syncytium suffers remarkable changes: picnotic nucleus, nuclear disintegration, thinness and interruption. Many of these vessels have exploited in the stromal region (Figure 4). If they are found in the stromal surface the erythrocytes are expelled to the intervillous space (Figure 5).

Increased numbers of villi are seen shaped by syncytium very thin which surround to numerous erythrocytes. With frequency a disorganized structure of the stem villi can to be observed which contain dilated vessels (Figure 6). These stem villi in some cases have a great deal coverture of fibrinoid deposition and in other cases the placental villi is seen buried by fibrinoid (Figure 7). X-cells can be seen in stem villi which are associated with fibrinoid deposition (Figure 8).

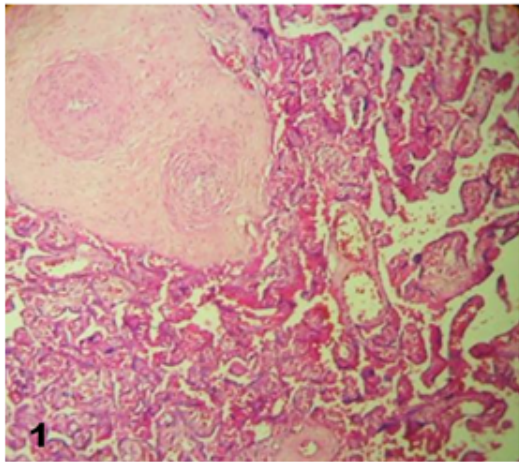


Figure 1: Closed vessels are observed in part of a stem villi associated with degenerative villi 400x H&E.

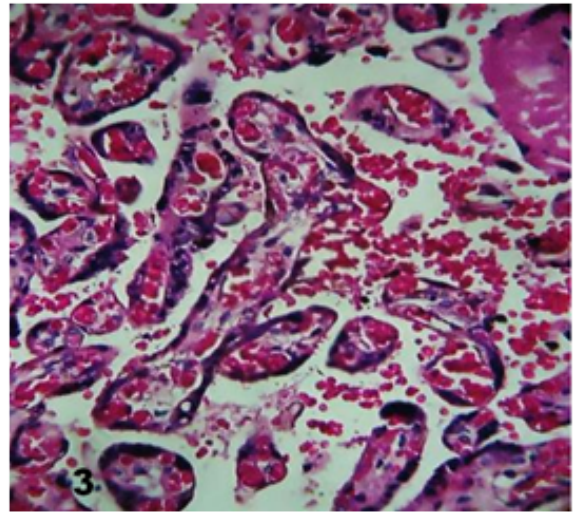


Figure 3: Placental villi with vessels dilated that occupy all the region stromal 400x H&E.

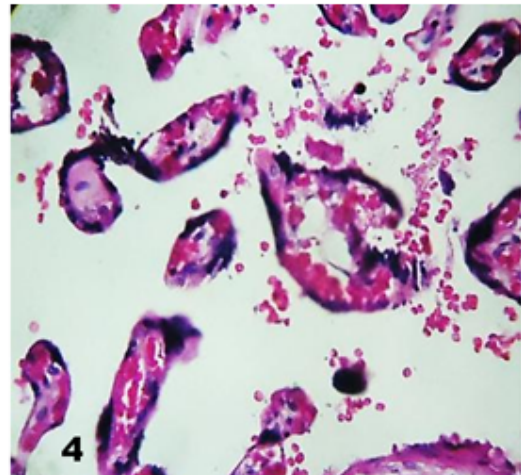


Figure 4: In stem villi dilated vessels have exploited in stromal region 400x H&E.

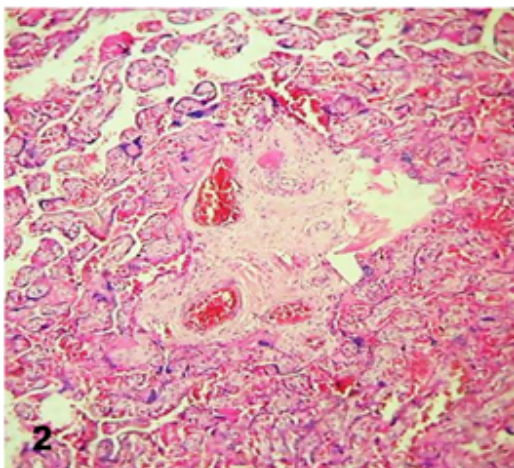


Figure 2: Stem villi contain a vessel with strictured muscular tissue and other opened seen in longitudinal section 400x H&E.

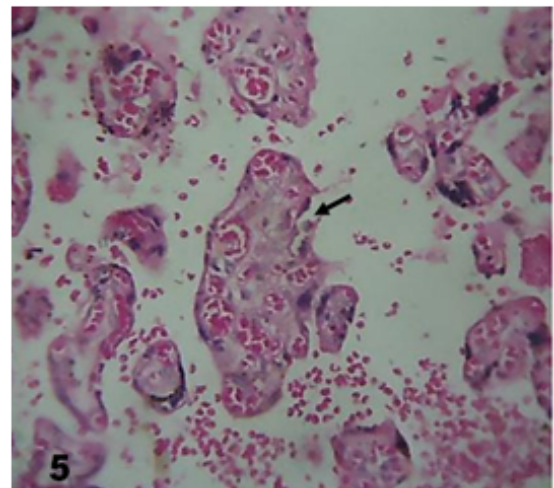


Figure 5: Vessel of stem villi (arrow) has expelled erythrocytes to the intervillous space 400x H&E.

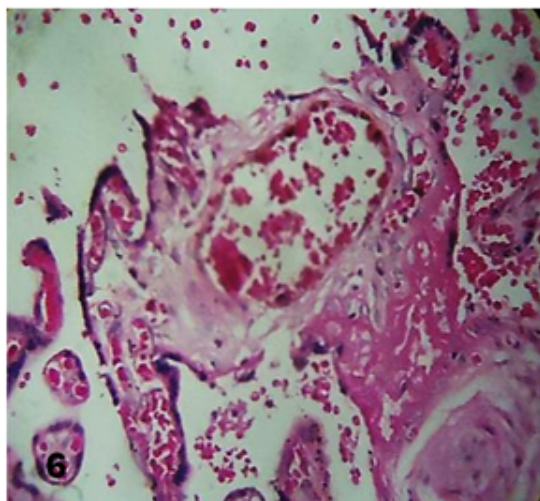


Figure 6: Stem villi is seen in disorganized structure 400x H&E.

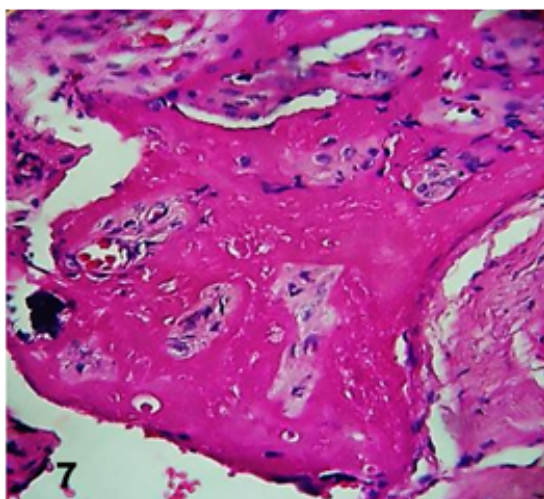


Figure 7: Ghost villi are found in fibrinoid deposition 400x H&E.

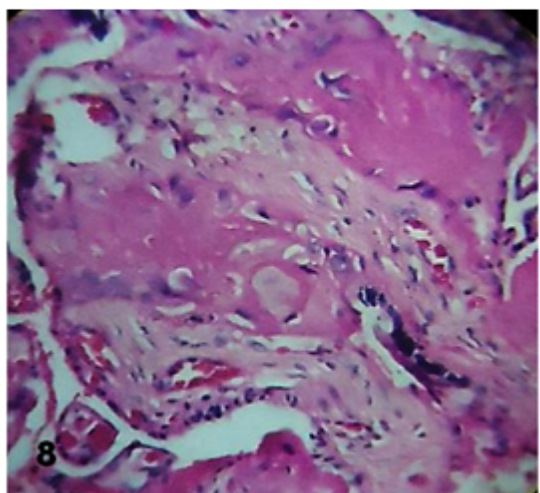


Figure 8: Fibrinoid deposition and x-cells are observed in stromal region of stem villi 400x H&E.

Discussion

In a previous study a general description of the placental villi in patients with high weight and prolonged pregnancy, whose chronic hypertension provoked severe degenerative changes to the placenta has been recently published [7]. The presence of hemorrhagic placental villi was seen in this paper. It has been recognized that hypertension provoke reduced utero placental blood flow [8]. In these cases narrowing of vessels has been demonstrated [9].

Closed vessels associated to chronic hypertension are indicative of the possibility that lead to the formation of thrombosis. This event provoke placental ischemia or low intravillous blood flow which could increase significantly the prostaglandins $\text{PGF}_{2\alpha}$ during vasoconstriction [10]. This abrupt diminution of the oxygen tension in this blood produce disorganization of the muscular media of vessels as has been seen in stem villus in placenta associated with obesity and hypertension [10].

The chronic hypoxias trigger tissue oxidative stress and increase apoptosis of the placental villi [11]. Micro particles of syncytiotrophoblast, fragments, rests, and micro villi from syncytium could be causing endothelial damage [12]. Damaged endothelium leak out plasma to muscular media provoking disaggregation of smooth muscle cells and their disorganization associated with the edema originated [3].

Long standing hypertension with severe elevation as can occurs in chronic hypertension can directly damage blood vessels producing severe necrotic damage contributing with the disappearance of villi. Chronic vasoconstriction and increased intraluminal pressure could lead to vascular obliteration through progressive mural hyperplasia.

In these cases associated with obesity this low blood flow can to be caused by deficient dilatation of utero placental vessels associated with sharp arteriosclerosis during hypertension. Fibrinoid necrosis is produced in this condition of low blood flow in the intervillous space and placental villi are found buried by fibrinoid deposition [7].

Blood vessel structure is altered in obesity with increase in vessel diameter, limiting caliber and distention of vessel walls, mechanisms that could affect placental blood vessel structure in maternal obesity [13]. Enhanced understanding of normal and aberrant placental structure of vessel in early pregnancy of obese woman is required [14].

Obesity has indicated increased placental vascularity or chorangiomas which would be an adaptable response to low blood flow in the intervillous space [15,16]. Notwithstanding vascular villi are found in 10.9% in severe hypertension and 7.27% in mild hypertension from placental lesions as infarctions and villous fibrinoid necrosis [17]. This reduction of vessels could to be being provoked by intravillous hemorrhagic here expressed.

The clear regions or zones of hyalinization that are seen in stromal region of stem villi correspond with the disappearance of these vessels [7]. So, hypovascularity of the villi has been reported in the hypertensive placenta [18]. Besides, a recent study about pathological study of placenta in pregnancy with hypertension and special reference to preeclampsia describes that hypo and hyper vascularity too were seen in toxemia of pregnancy [19].

It has been described that elevated angiotensin II may favors vessel constriction in placental chorionic villi and contribute to impaired fetal blood flow and decreased fetal nutrition observed in a hypertensive disorder as preeclampsia [20].

Chronic hypertension has been associated with an increased rate of vascular placental maternal bad perfusion [21]. Careful medical attention is necessary in these cases of obesity and hypertension since fetal stillborn can to be the result of post delivery [22].

Conclusion

In conclusion, during chronic hypertension patients with obesity present intravillous stromal hemorrhage in the placental villi which could to put in danger the fetal good development with decreased fetal nutrition.

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